SACHS (B.)

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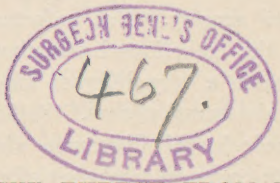

WHAT CAN WE EXPECT FROM THE SURGICAL TREATMENT OF EPILEPSY?*

By B. SACHS, M. D.,
PROFESSOR OF MENTAL AND NERVOUS DISEASES IN THE NEW YORK POLYCLINIC.

The treatment of so grave a disorder as epilepsy is a subject which may well claim the attention of all medical men. The disease is a veritable scourge that leaves its indelible mark upon the victim, often attacking him at an early age, unfitting him for the serious work of life and blighting all the hopes dependent upon him. In view of the importance of the subject, it need not surprise us that the subject has come up for discussion so frequently before our learned societies; and no apology is needed, I believe, for continuing before this Academy a discussion which was conducted most ably only a few weeks ago before the American Surgical and Neurological Associations at Washington.

The brilliant achievements of American and European surgeons have given us the utmost confidence in the possibilities of cerebral surgery, and, with the increasing knowledge of neurologists in localizing accurately the site of disease in the brain, there would seem to be no good reason

* The opening paper in a discussion on the medical and surgical treatment of epilepsy, held before the New York Academy of Medicine, October 15, 1891.

why the results of cerebral surgery should not equal those in abdominal surgery. But, unfortunately for the patient and for us, the complicated structure of the brain makes cerebral disease a very different affair from disease in or around the abdominal viscera. Sufficient allowance is not, as a rule, made for this difference, whence it follows that in the case of the surgical treatment of epilepsy much has been expected and but little has been realized. It seems wise to me, therefore, before we allow our hopes undue scope, that we should stop to inquire what we can expect from the surgical treatment of epilepsy.

To those who have not acquainted themselves with the literature of the subject a simple answer may occur. They may say: "Take the recorded cases, note the results, and make your inferences"; but in the case of the surgical treatment of epilepsy statistics are useless. A few successful cases have been reported, and even these with undue haste. Von Bergmann * and the latest author, Sahli, † have given up the attempt to tabulate the results of operations upon epileptic patients. I started out on the same path a few months ago, but soon found it would be love's labors lost. In this matter of the cure of epilepsy after operation the memory of medical men is not as reliable as it fortunately is with regard to most other diseases. A distinguished surgeon stated recently before one association that he knew of a case of traumatic epilepsy in which the cure was of ten years' duration, and before another society, a few days later, that the longest cure that he knew of was of three years' duration. But what of the innumerable failures? They have not been reported with the same candor

* Von Bergmann. *Die chirurgische Behandlung von Hirnkrankheiten.* Berlin, 1889.

† Sahli. Volkmann's *Samml. klin. Vortr.*, Vortr. No. 28. N. F., 1891.

and promptness with which most men have heralded their short-lived successes. And if the attacks are inhibited for a number of weeks or months, is even this temporary success to be put to the credit of the operation? As long ago as 1875 Maclaren* insisted that epileptics were improved by any operation whether it be for cancer of the pelvic organs, for joint disease, or what not; and it is as likely as not that some of the supposed cures after ovariotomies may be accounted for in this way, though I am not willing to go to the extreme to which Dr. White,† of Philadelphia, has recently gone, in putting the improvement following trephining for traumatic epilepsy in the same category with the "curative effects of operations *per se*," simply because no gross organic changes were found in the organ so exposed.

I propose to answer the question embodied in the title of this paper by presenting to you the views I am forced to hold regarding the nature of the disorder which we attempt to cure, and by giving you the results of my own experience in the surgical treatment of epilepsy—an experience that I owe chiefly to the kind offices of Dr. Gerster and Dr. Wyeth, with whom I have been associated in fourteen cases of cerebral operation, ten of which were done for the relief of epilepsy.

Let me remind you that epilepsy is a symptom, not a disease; that it is often merely one of a number of symptoms pointing to organic disease of the brain—to tumor, hæmorrhage, abscess, or widespread meningitis and sclerosis. In other cases it is a direct or remote effect of traumatic injuries to the skull or brain. In addition to these we have cases of genuine epilepsy, so called. Some one has suggested that it would be better to call them cases of

* Maclaren. *Edinb. Med. Journal*, January, 1875.

† White. The Supposed Curative Effect of Operations *per se*. *Annals of Surgery*, August and September, 1891.

epilepsy from unknown cause, and our modesty should, I think, incline us to this latter view. For one, I find that I see relatively fewer cases of genuine epilepsy than I did in former years. On closer examination I have not infrequently found that the epilepsy had taken its start from a long-forgotten injury or accident; that it was in its earlier days associated with paralysis, the paralysis having left but the slightest traces, while the epilepsy has remained distinctly enough. Nor is it to be supposed that a true epilepsy—I do not mean single convulsive seizures—is a functional disease. In our ignorance we may call it so, but with the improved methods of examining cortical tissue I am confident that we shall before long be able to demonstrate its anatomical substratum. Two French authors have been leading the way in this inquiry. Marie,* who goes to the extreme of denying hereditary epilepsy, claims that children may be born epileptics, but they have not been conceived as such. By which he implies that, as in the case of congenital cerebral palsies, some slight lesion has been established during the intra-uterine period; that this lesion may be lost sight of, but the secondary degeneration following upon it is the cause of this supposed hereditary epilepsy. Féré † has furnished strong evidence in favor of this view. He induced Chaslin ‡ to examine five brains taken from epileptic subjects; in all of these brains the most careful microscopical examination revealed an increase of neuroglia tissue with the formation of small fibrils emanating from the spider cells of the neuroglia. Chaslin infers that this is a sort of gliomatous sclerosis, and that this sclerosis is to be found in epileptic brains of entirely normal macroscopical appearance.

* Marie. *Progrès méd.*, 1887, No. 44.

† Féré. *Les épilepsies et les épileptiques*, Paris, 1890.

‡ Chaslin. *Semaine méd.*, 1889.

I have led you into this discussion of the anatomy of so-called genuine epilepsy in order to have you associate in your minds the idea of secondary sclerosis of the cortex with epilepsy. This secondary sclerosis is the pivot upon which the entire question turns. I repeat that in cases of idiopathic epilepsy the initial causal lesion has not yet been determined; idiopathic cases have by common consent not been considered proper cases for operation; but we have learned to operate in cases of localized epilepsy —Jacksonian epilepsy—due to focal disease, whether of traumatic origin or not, and in these cases the focus of disease is present, and so is the secondary sclerosis. If years pass by before death ensues, the focal disease may be beyond recognition, but the secondary sclerosis can be and has been recognized. In early childhood the cortex suffers focal injuries which might well be called traumatic, if they were due to an external force; but, whatever the cause may have been, we have a meningeal hæmorrhage often enough; the clot resting upon the cortex may be absorbed, but it has given rise to a local change which extends by degrees until it finally leads to secondary changes in the form of a lobar sclerosis. Paralysis and epilepsy are the chief symptoms of this condition. Instead of a meningeal hæmorrhage due to difficulty during labor, imagine a traumatic hæmorrhage; disregard the ætiological differences and the two accidents will result in the same processes.

Let us push the argument one step further. Every one is familiar with the fact that the convulsive seizure of a localized type is due to an irritative lesion of the motor or sensory centers. The most familiar cases of localized epilepsy are those due to discharging lesions in the motor areas. It is well known, too, that convulsive movements of a thumb or an arm point to a discharging lesion in the center or centers representing these parts. Those parts only are capable

of a discharging lesion which are not actually destroyed; if destroyed, we have absolute paralysis and not localized convulsive seizures. Does the diseased area contain within itself the irritating power, or is this irritation conveyed to it from other parts? As a tumor grows near a center, it irritates that center and causes convulsive seizures; as soon as it has displaced or destroyed the center, the formerly convulsed part becomes paralyzed. The injuries and morbid processes with which we are here concerned rarely lead to the destruction of a center; it is capable of discharging, and the irritation it needs seems to me to be supplied by that secondary sclerosis of which it—the focal injury—has been the prime cause. Is there any clinical evidence that the secondary sclerosis plays this part? I shall be very much mistaken if it does not explain the very curious fact that in traumatic injuries and in the cortical diseases of early childhood the epilepsy does not, as a rule, develop for months and even years after the focal lesion has been established. It takes months, and even years, until a large amount of sclerosis is established. It is fortunate that focal injuries are not invariably followed by sclerosis; why it should develop in some cases and not in others it is difficult to say; the severity of the lesion is not the sole determining factor.

Some may maintain that, while this reasoning may apply to epilepsy following organic diseases, the sclerosis has not been actually demonstrated in cases of traumatic injuries. The fault is with the investigator. Bergmann* reports the case of a man who had received a gunshot wound of the left parietal bone in 1870; he was operated upon and bone was removed. Two years later he had the first epileptic attack. These attacks continued for fourteen years; then Bergmann trephined over the scar. The patient did well for one month; after that he fell into the status epilepticus, in

* *Loc. cit.*, p. 160, second edition.

which he died. The author assures us that the wound had healed perfectly, and that the cortex and dura were entirely normal—macroscopically, it may be—but no microscopical examination was made. Inasmuch as some of the cases to be operated upon hereafter may die, it will be well to make a most careful histological examination of the cortex in such cases.

Returning to the practical bearing of these pathological studies, we infer that we have an initial focal lesion and a condition of secondary sclerosis to deal with. It is our plain duty, therefore, to prevent the development of secondary sclerosis if possible, or, if it has been developed, to neutralize its effects. The first part of our task is by far the more difficult. Not knowing as yet the exact conditions under which this sclerosis is developed, we can scarcely be expected to meet these conditions; but we can attempt to diminish the initial lesion and, if possible, to remove it. This is equivalent to a plea for early operations in traumatic and organic cases. How can we neutralize the effects of a well-established sclerosis? Shall we say by excising the diseased area? The method is rational enough, since we can not rid the brain of the sclerosis.

This method of excision has been applied by Horsley,* Keen,† Bergmann, and others. The results have been satisfactory in some cases, disappointing in others. In spite of the removal of the center, the sclerosis has exerted its power through other channels, through other irritable centers. If the diseased center is the only irritable area, the result will probably be a good one; but we have no means of predicting whether or not this will be the case. And to make matters worse, excision of a center means loss

* *Brit. Med. Journal*, April, 1887.

† Keen. *Am. Jour. of the Med. Sciences*, October and November, 1888.

of function. You may not cure the epilepsy, but you are very apt to paralyze the convulsed part; but this function of the excised part is very apt to be assumed by other parts of the brain, particularly in young persons, and among older persons the patient, if left to make his choice, will prefer a local paralysis to a severe epilepsy.

The practical conclusions to be drawn from the foregoing are these:

1. In a given case of traumatic or organic lesion, operate as early as possible, to prevent the development of secondary sclerosis.

2. If you have not operated at the outset, the onset of epilepsy is a warning that secondary sclerosis has been established; by operation at this time you may avoid an increase of the trouble.

3. Excision of the diseased area is the only rational operation; if all other centers are not in an irritable condition, the operation may be thoroughly successful.

But if we can not easily cure epilepsy, we may improve the patient's condition by diminishing the number of attacks. What we can accomplish I propose to discuss in the second part of this paper. I refrain purposely from entering upon the subject of operative interference in cases of tumor or abscess of the brain, as the advisability of operating is governed by motives other than the cure of the epilepsy. Traumatic epilepsies will attract us first, and next I wish to enlist your interest in certain forms of epilepsy associated with infantile cerebral palsies. These latter diseases have been so generally overlooked that the epilepsy constituting one of the symptoms has rarely been recognized as a special form.

Traumatic cases call for immediate surgical interference. Whenever the skull has sustained a severe or even a moderate injury, a surgeon or the attending physician

should do an exploratory operation to make sure that there is no depression of bone. As trepanation is not a very dangerous operation, it would be better to do this than to have the slightest doubt. I was pleased to note that Dr. Agnew * insisted strongly on this point in his recent paper at Washington. Together with Dr. Wyeth, I had the privilege of seeing, only a few weeks ago, a robust individual who had sustained a fracture of the skull by falling from his wagon; he was picked up in an unconscious condition, and, with the exception of an occasional lucid moment, remained in a condition of stupor up to the time of the operation. There was no paralysis and no focal symptom of any sort. Yet during the exploratory examination a large fracture was found which extended well back from the coronal to the lambdoidal sutures, and running for a part of its course along the sagittal suture. A trephine opening was made to see whether there was any splintering of the inner table, or whether the dura had been lacerated. As far as we could see, no severe injury had been inflicted; and the good, but slow, recovery which the patient has made may possibly have come about in this special case without any operation. Yet these are the very cases which so often develop epilepsy from pressure of depressed bone; and it seems to me fully as important that the surgeon should operate early to determine whether there is a depression as that he should defer operation until epileptic symptoms appear which may place the case beyond the possibility of surgical relief.

After the epilepsy following traumatic injury of the skull or of the brain has been developed, there is still hope that the epilepsy may, in a few instances, be inhibited by surgical methods. For ages past trepanation has been the classical operation in these cases. Wherever depressed

* Agnew. *University Med. Magazine*, 1891.

bone presses upon any part of the cortex, or an old scar acts as a source of irritation, the removal of such bone or scar is clearly indicated; in many cases improvement, if not a cure, of the condition follows. We must seek an explanation, however, for the improvement which follows trepanation in many cases of traumatic epilepsy of long standing in which there is no old scar and no marked depression of bone. We will not explain this on the theory that any or every operation helps; we know, however, that adhesions between the brain and its coverings are apt to be formed, and that traumatic cysts are very frequent. The trephine opening may therefore relieve the increased pressure due to these morbid conditions. It will be well to enlarge the trephine opening and to make it as ample as the conditions will permit. To show you how much or how little may be accomplished by mere trepanation, allow me to present the very condensed histories of a few cases of traumatic epilepsy. The full histories I hope to publish in due course of time, in conjunction with the surgeons who have done the operations.

Case I.—Boy, aged sixteen years, from Madison, Wisconsin; no hereditary history. At the age of eighteen months fell out of a first-story window; since that time epileptic attacks of great severity, with occasional intervals of freedom from attacks. These generally begin in the right arm and extend to the leg and face; often they become general. Loss of consciousness in all attacks. I referred him to Dr. Gerster. Operated upon February 23, 1891. The skull was trephined over the arm area, which was proved to have been exposed by the electric test; the trephine opening extended so as to expose the greater part of arm and leg centers. Attacks set in two days after the operation and have continued with old-time severity. Result, no improvement. The father would not consent to a second operation.

Case II.—Young man, aged twenty years; works on his

father's farm in New Jersey. At the age of twelve years was pushed backward over the pole of a wagon, striking, according to account, on the back of his head; was unconscious for a few minutes, but went to work. A week later the first general epileptic attacks; these attacks had recurred at varying intervals for two or three years. For the past three years the boy had attacks of typical Jacksonian epilepsy, in which the muscles about the right half of the mouth only were convulsed. I had occasion to see several such attacks. These minor attacks would occur many times a day. This series would be interrupted by an occasional severer attack of localized convulsions, ending up in general convulsions, loss of consciousness, etc. The boy, who is a fairly bright lad, desired the operation. This was done February 13, 1891, by Dr. Gerster. As demonstrated by the faradaic stimulation over the exposed area, the trephine opening had been laid exactly over the center representing, according to Horsley,* the upper face and angle of the mouth. Adhesions were found under the button of bone which was removed. Good recovery, but attacks set in within a week after operation, and in these attacks the eyelids were convulsed, showing a slight extension of the focal lesion. A few weeks later the attacks were as of old in every respect. "No improvement whatever," so the boy reported to me eight months after the operation.

Somewhat better success has followed upon operation in two cases of sensory epilepsy of traumatic origin; as they are rare forms of epilepsy, I wish to refer to them briefly in this connection.

Case III.—J. D., aged eight years. When seven months old fell upon the left side of the head; at eleven months began to have epileptic convulsions; twitchings usually began in the right arm and leg, and the mouth would be drawn to the left side. After the age of five years these attacks disappeared. Since last March the boy has had similar attacks; but with the onset of these attacks it was noticed that the ear began to discharge. The convulsive attacks were regularly preceded by auræ, in

* Horsley. *Am. Jour. of the Med. Sciences*, 1887, vol. i.

which he would either perceive a very foul odor or else imagine a steam-car close upon him. He would close the window to keep out the noise, and at once fall into a convulsion. The attacks would be more frequent whenever the discharge from the ear ceased. About one such attack occurred every week before operation.

The mastoid region was not painful, but everything pointed to mastoid trouble, and hence I sent him to Dr. Gerster for operation. On August 14th Dr. Gerster chiseled through the mastoid process into the middle ear and removed several sequestra; no pus. The wound healed quickly. The boy has had but one attack in two months, and is far less irritable and brighter than before.* It is interesting to note that with the onset of this ear trouble the old epileptic tendency due to traumatism had been revived.

Case IV.—A man, aged thirty years, of alcoholic habits, at the age of ten years was struck by a stone on the back and side of the head. Four years later he began to have general epileptic convulsions. These continued regularly; every two weeks he would have two or three attacks in a single day. Patient has become stupid and irritable; has left lateral hemianopsia. Operation, July 24th, by Dr. Gerster. Removal of occipital scar; external plate found to be depressed; this was removed with chisel. After dura was exposed and adhesions were cut away, the opening in the occipital bone was enlarged to the size of a silver dollar. The wound did well. Sixteen days after operation three epileptic attacks occurred; they were followed by transitory delusions of persecution, from which he soon recovered. He was discharged on August 15th, and had no attack until October 1st, after taking a large amount of liquor for the relief of pain. The result may be designated as a marked improvement. The hemianopsia has remained unaltered.

This is an epilepsy starting from one of the sensory areas of the brain, but whether the patient actually had a visual aura he is too stupid to state.

* A second operation was done about two months later, since which time the boy has been free from attacks.

Our own experience in this matter is not unlike that of other physicians. Horsley, Bergmann, Park, Keen, and others have not fared much better.

In the search after better methods, Horsley's suggestion, in case of focal lesion to remove the entire diseased center, met with general favor. With the aid of our present methods of determining centers, and particularly if we allow the result of faradaic stimulation of an exposed area to be our chief guide, we can very accurately determine the extent of the area to be removed, and I have satisfied myself that this can be done before the dura is opened. But, as I have intimated before, even the excision of diseased centers is not an unmixed good. First of all, it often fails. Attacks have returned after such operation in the cases of Bergmann, Park,* and Keen. In one of Park's cases the contents of the cyst were emptied, but the cyst was not removed. Horsley's first case has been the only very positive success, no attacks having occurred for twenty-two months after operation.†

Rational as this method seems to be, there are reasons for its failure. The entire center may not have been removed; the paralysis of the convulsed part has been thought by Keen and others to be proof of the fact that the entire center has been removed. Then, again, if an arm center has been the actual focus of disease, the neighboring face or leg center may, in the course of years, have attained a sufficient degree of irritability to become the discharging center, and, furthermore, the existing sclerosis will help to advance this or other centers to the dignity of a discharging center, if they have not already acquired this unfortunate function.

* Park. *N. Y. Med. Jour.*, November, 1888.

† I can not find any later reference to this case. Other cases have been reported, but before a sufficient period of time had elapsed.

Another serious feature of this excision operation, and indeed of all cerebral operations, is the possibility that the operation itself may lead to the formation of cicatricial tissue in or around the cortex, which may do more damage than the initial lesion. Yet, from what I have seen of the condition of brains years after an operation, I believe this danger from new cicatricial tissue to be quite slight.

Granting the entire success of the operation in question, you have at best in many cases substituted a paralysis for an epilepsy. In the case of an arm or face center, most patients would consent, and prefer the paralysis to their epileptic seizures. Few would care to have their leg centers excised, and to wait until some vicarious center may give them power to walk; and I doubt whether in Case IV, which I reported above, the patient or my own conscience would have permitted me to remove one visual center. Excision of a center, while it promises relief in a few well-selected cases, is not so much of a divine inspiration as it appears to some to be. Let us prevent traumatic epilepsy as far as in our power lies; it will be easier to prevent it than to cure it.

I consider it my duty to call your attention to the epilepsy associated with the cerebral palsies of children. In a paper * published last year it was shown that forty-four per cent. of all cases of infantile cerebral palsies develop epilepsy, and I have stated that there could be but little doubt that, of all cases of ordinary epilepsy, a very fair proportion were developed in connection with infantile palsies. This view has been accepted by later writers. I have seen at least half a dozen cases of epilepsy within the past year which were supposed to be cases of idiopathic epilepsy, but which, when examined carefully, revealed the traces of an

* Sachs and Peterson. *Journal of Nervous and Mental Disease*, May, 1890.

old hemiplegia. Nor are the pathological conditions underlying these palsies properly recognized.

One case of Horsley's, a boy four years of age, with right hemiplegia, who had as many as thirteen or fourteen attacks per day, is a case in point. Dr. William A. Hammond * some time ago reported the case of a girl of twenty, afflicted with an old hemiplegia, in whose brain he found a large cyst which was evidently the leavings of a former hæmorrhage, and Case V of Dr. Keen's † latest paper is not merely a case of defective development, but one of infantile cerebral hemiplegia with epilepsy and idiocy. I would not call attention to this class of cases if we did not find among them the only cases of non-traumatic epilepsy which demand surgical interference.

These palsies come on either in the intra-uterine period or early in life. The initial lesion in the acquired cases is generally a hæmorrhage, thrombosis, or embolism, and this focus of disease leads in many cases with rather surprising rapidity to the development of secondary sclerosis throughout the cortex. In fully ninety-five per cent. of all these cases the lesion is in or upon the cortex. The lesion—a hæmorrhage or softening, say—is very apt to be strictly limited to one or more areas. It need not, therefore, surprise us that typical Jacksonian epilepsy is found in some of these cases, or that we find scars and cysts and sclerosis just as we do in the traumatic cases.

The three cases of this class which I shall refer to were operated upon during the past year—two of them by Dr. Wyeth and one by Dr. Gerster. The histories are briefly as follows:

Case V.—L. C., male, aged six years. Onset of disease at the age of five years with right hemiplegia and convulsions of a

* Hammond. *N. Y. Med. Journal*, August, 1890.

† Keen. *Am. Jour. of the Med. Sci.*, September, 1891.

Jaksonian type which have been repeated every week since, finally increasing to as many as five daily; athetoid movements of right hand; is irritable and bad-tempered. Operation was done December 29, 1890. A large opening was made in the skull over the motor arm area of left hemisphere and enlarged from this; adhesions to skull broken up. No attacks for six weeks after operation, and none for several months after leaving the hospital. I have not heard from the boy in fully three months.

Case VI.—A girl, aged sixteen, who has had right hemiplegia and epilepsy since early childhood; epileptic attacks very frequent and affecting paralyzed part exclusively. Dr. Wyeth operated upon the girl in the Polyclinic Hospital, exposing, by the method which I must leave to him to explain, the entire left motor area. In this area I determined by the use of the faradaic current the exact arm center. The dura was opened in semicircular fashion, but, as the entire area seemed normal, we did not at the time excise any part of it. The dura was closed, and the wound healed nicely; she was free from attacks for at least five months.

Case VII has been by far the most interesting.* I was asked by Dr. Wyeth to see A. H. G., aged thirty-two, who had applied to him for the relief of his epilepsy. The history showed that the epilepsy had been developed at the age of two years, and that with the epilepsy a complete left hemiplegia appeared. On examining the patient this hemiplegia was evident enough, and this, taken in conjunction with the history of unilateral attacks, led me to look for a focal lesion—a hæmorrhage probably—which, from the nature of the attacks, I thought likely to be most marked in the arm center. Dr. Wyeth trephined over this site, enlarged the opening, cut open the dura, and then exposed the discoloration and thickening of the pia which was adherent to the cortex beneath. A number of incisions were made into the cortex at this point, breaking up old adhesions, and lessening the increased tension at this point. The patient did well after the operation. On the second day the paretic arm was

* This case has been referred to in the author's paper on the Pathology of Infantile Cerebral Palsies, this journal, May 2, 1891.

paralyzed, but after the lapse of a few days it was no worse than before the operation, while the epileptic convulsions, which had appeared at least once a week before the operation, did not occur until six months after the operation. The further course of this epilepsy will have to show whether any permanent improvement has been gained.

Simple trepanation seems to be more successful in these epilepsies associated with infantile cerebral palsies than in the traumatic forms, probably because of the still greater frequency of cysts in these diseases than in the traumatic epilepsies. The early recognition of these troubles is of great importance; and the question naturally arises whether we can diagnosticate the lesion with sufficient accuracy to encourage the surgeon to operate at an early day before secondary degeneration is established. I believe this will be possible in many cases, but the disease sets in frequently at a very tender age at which cerebral operations are but poorly tolerated; moreover, the epilepsy, although a probable sequel, is still a remote contingency; the paralysis represents the reality, and parents will be most apt to tell the physician to care for the present only, more particularly if looking to the future means a possible increase of the paralysis. As soon as epileptic symptoms appear, the paralysis has the value of a focal symptom; the centers should be exposed, and if not removed, they should at least be treated in accordance with the special indications of the case. In children, excision of a center is a less serious affair than in the adult, for in the former other parts of the cortex are capable to a greater degree of assuming the functions of the destroyed part. I am confident that, if these cases of infantile cerebral palsies are more generally recognized, and if we succeed in checking the tendency to epilepsy in them, the total number of epileptics will be noticeably diminished. If the surgical treatment of epilepsy be of any value at all,

it is in view of the foregoing not to be restricted to the traumatic forms, but let it be applied also to those epilepsies which are associated with the cerebral palsies of childhood.

In conclusion, I would say that, under favorable conditions and by the methods described in this paper, the surgeon may be able to cure a few cases of epilepsy. He will be able to improve many, but surgeons and neurologists should in future make an earnest effort to *prevent* epilepsy.

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